ABNORMAL GLUCOSE-TOLERANCE TEST IN THE NATAL INDIAN AND AFRICAN HYPTERTENSIVE PATIENT*

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Hypertension is a common disease in the Natal Indian and African patient; diabetes too is common, as evidenced by the large number of patients who attend the Diabetic Clinic. This study was begun because at the Hypertension Clinic, glycosuria was detected in many patients who had not been referred from the Diabetic Clinic. All the patients were urban.

METHODS

Only those patients who, in addition to a high resting diastolic pressure (> 120 mm Hg), showed evidence of hypertensive changes in the fundi, heart or kidneys were included in this series. It was not necessary to have the patients on a high-carbohydrate diet as their normal diet is generally high in carbohydrates. Venous blood was sampled in the fasting state and one and two hours after a 100-G glucose load. Glucose levels were measured using the Technicon Auto Analyzer. Nearly all the patients had a 12-lead electrocardiogram (ECG) done at rest, blood urea estimations and a radiograph of the chest. All had been on thiazides at some stage, and this was usually for an average period of 3 years, extending up to 5 years.

RESULTS

Interpretation of the modified glucose-tolerance test (GTT) was based on the criteria of Conn and Fajans who considered abnormal, a blood-glucose level of 160 mg./100 ml. or greater after one hour and 120 mg./100 ml. or greater after two hours. The reason for taking Conn and Fajans' criteria was to have uniformity with a similar study by Nye. Because determinations were performed on plasma, using the Technicon Auto Analyzer, these levels were converted using a formula based on a method described by McDonald et al. These levels (similar to those of Conn and Fajans) were 190 mg./100 ml. after one hour and 145 mg./100 ml. after two hours of a 100-G load of glucose.

TABLE I. INCIDENCE OF ABNORMAL GTT IN INDIAN AND AFRICAN HYPERTENSIVE PATIENTS

<table>
<thead>
<tr>
<th>Type</th>
<th>Diabetic</th>
<th>Non-diabetic</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indian hypertensive</td>
<td>104 (78%)</td>
<td>29</td>
<td>133</td>
</tr>
<tr>
<td>African hypertensive</td>
<td>50 (49%)</td>
<td>51</td>
<td>101</td>
</tr>
<tr>
<td></td>
<td>154</td>
<td>80</td>
<td>234</td>
</tr>
</tbody>
</table>

Incidence of Glycosuria in Indian and African Hypertensives

Glycosuria occurred in 58% of 133 Indian hypertensives and 24% of 101 African hypertensives. This is a highly significant difference. The prevalence of glycosuria in diabetic Indian hypertensives was significantly higher than in non-diabetic Indian hypertensives, i.e. 66% of 104 and 33% of 29 cases respectively. Diabetic African hypertensives had a significantly higher rate of glycosuria, i.e. 45% of 90 patients compared with 11% of 51 patients in non-diabetic African hypertensives. From this study there was a fair percentage with a low renal threshold for glucose.

Religion of Indian patients: Of the 104 patients with diabetes, 94 were Hindus and 10 were Moslems. Of the non-diabetic Indians, 26 were Hindus and 3 were Moslems. However, it must be remembered that the ratio of Hindus to Moslems in Durban is about 5:1 (population census 1960) and that Hindus, being the poorer of the two religious groups, would attend this hospital more often.

Table II shows that the sex distribution among the Indian diabetics was equal, whereas more of the African diabetics were females. Table III shows that the majority of the patients had a relatively normal blood urea and that uraemia played little part in influencing the GTT as has been determined by Neubauer and Linder et al.

<table>
<thead>
<tr>
<th>Type</th>
<th>Diabetics</th>
<th>Indians</th>
<th>Africans</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>51</td>
<td>19</td>
<td>31</td>
<td>84</td>
</tr>
<tr>
<td>Female</td>
<td>53</td>
<td></td>
<td>31</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>104</td>
<td>50</td>
<td>154</td>
<td></td>
</tr>
</tbody>
</table>

Table II shows that the majority of the patients had grade 1-2 retinopathy, while the findings in Table V show that the majority of the patients had a relatively normal blood urea and that uraemia played little part in influencing the GTT as has been determined by Neubauer and Linder et al.

Mean blood pressure (diastolic) in diabetics and non-diabetic patients (African and Indian) showed no
significant difference between any 2 of the 4 groups: t test p > 0.05.

TABLE V. DISTRIBUTION OF BLOOD UREA IN MG./100 ML.

<table>
<thead>
<tr>
<th>Type</th>
<th>No. of cases</th>
<th>&lt;30</th>
<th>30-40</th>
<th>40-50</th>
<th>50-100</th>
<th>100-150</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetic hypertensives</td>
<td>154</td>
<td>57</td>
<td>50</td>
<td>21</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Non-diabetic hypertensives</td>
<td>80</td>
<td>35</td>
<td>40</td>
<td>3</td>
<td>2</td>
<td>0</td>
</tr>
</tbody>
</table>

Family History

Glucose-tolerance tests were not done in the families. A family history of diabetes mellitus in diabetic Indian hypertensives and diabetic African hypertensives showed a significantly higher incidence of diabetes mellitus in relatives of diabetic Indian hypertensives compared with diabetic African hypertensives, i.e. 15% of 104 cases and 2% of 50 cases respectively. A family history of hypertension in diabetic Indian hypertensives compared with diabetic African hypertensives occurred in 38% of 104 cases and 8% of 50 cases respectively. This was a significant difference. There was no difference in the family history between diabetic Indian hypertensives and non-diabetic Indian hypertensives, or between diabetic African hypertensives and non-diabetic African hypertensives.

The possible reasons for the marked disparity in the family histories between the Indian and African patients could either be due to the fact that the African has only recently become more aware of Western medicine and with it medical terms like diabetes and hypertension, or else diabetes and hypertension are new diseases emerging in the African population.

Electrocardiogram Comparisons

Electrocardiogram changes of Q waves, left bundle-branch block, ST depression or a history of angina pectoris were studied in the patients. These changes were regarded as evidence of myocardial ischaemia. Evidence of myocardial ischaemia occurred in 18% of 95 Indian diabetic hypertensives compared with 4% of 25 Indian non-diabetic hypertensives. Though there were not enough patients to analyse in the group, there seemed to be a higher incidence of myocardial ischaemia in Indian diabetic hypertensives compared with Indian non-diabetic hypertensives.

There was a significant difference between the incidence of myocardial ischaemia in diabetic Indian hypertensives (18% of 95 cases) and diabetic African hypertensives (25% of 37 cases)

There was no significant difference in T wave inversion between diabetic Indian hypertensives and non-diabetic Indian hypertensives or in diabetic African hypertensives and non-diabetic African hypertensives. There was also no significant difference in ST deviation between diabetic African hypertensive patients and non-diabetic African hypertensive patients.

Potassium Study

Further and complete studies of all the patients with hypertension is being undertaken, but preliminary studies have shown (Table VI) that there was no difference in the potassium levels between diabetic and non-diabetic patients.

TABLE VI. POTASSIUM LEVELS IN DIABETICS AND NON-DIABETICS

<table>
<thead>
<tr>
<th>Type</th>
<th>Serum K below 3-5 mEq./l.</th>
<th>Serum K 3-5-5-0 mEq./l.</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetics</td>
<td>11 (16% of total)</td>
<td>58</td>
<td>69</td>
</tr>
<tr>
<td>Non-diabetics</td>
<td>6 (16% of total)</td>
<td>31</td>
<td>37</td>
</tr>
</tbody>
</table>

DISCUSSION

The incidence of an abnormal GTT in this series was 78% in Indians and 49% in African patients. One may ask if impaired tolerance for carbohydrates is more frequent in the hypertensive than in the non-hypertensive patient. Drazin found grossly impaired tolerance in 11 (21.1%) of 52 non-obese hypertensive patients; patients known to be diabetic were eliminated from this study. In the Japanese diabetes detection survey, Kobayashi found the frequency of diabetes mellitus to be 15% in hypertensive patients over the age of 40 and 3.5% in non-hypertensive patients in the same age-group.

There is as yet no complete study of the incidence of diabetes in the Natal Indian and African. In Durban, Woods estimated that the incidence of diabetes in an Indian sub-economic housing scheme was 4% in the 40-49 age-group and 14.8% in the 50-59 age-group. Walker and Seftel in Johannesburg found that the incidence in middle-class Indians was 10% in the 40-49 age-group and 16% in the 50-59 age-group.

Wilkins and Freis first showed that thiazides caused hyperglycaemia in some of their patients. Samaan et al. suggested from their studies that these agents caused a low rate of insulin production as a result of a direct inhibitory action on the islets of Langerhans. Shapiro et al. found that whereas the glucose metabolism became abnormal in some patients with mild diabetes or prediabetes who were treated with thiazides, there was no change in those with normal glucose handling initially. From the studies of Wolff et al. and the report to the European Society for the study of Drug Toxicity an editorial in the Lancet under the title 'Drug-induced diabetes', stated: 'the incidence of diabetes among treated patients in about 30% is disturbing but inconclusive'. However, even if one allows for the 30% incidence from thiazides in my studies, one is still left with the high proportion of 48% of abnormal GTTs in the Indian hypertensive patient.

From this study there seemed to be a higher incidence of myocardial ischaemia among Indian diabetic hypertensives than in Indian non-diabetic hypertensives. In the over-all pattern there was a higher frequency of myocardial ischaemia among Indian diabetic hypertensives compared with African diabetic hypertensives. Nye, in a study of 107 hypertensive patients, found that there were 35% with an abnormal GTT in those with coronary artery disease, compared with 16% with an abnormal GTT in those patients with no symptoms.

In the Natal Indian, McKechnie and Davidson found that the incidence of diabetes mellitus associated with coronary artery disease was 66%. Only one Indian hypertensive in this series had myocardial infarction, but it is
difficult to correlate this finding as the patients have not been followed-up for a long enough period.

Gardner et al. found that in rats, severe potassium deficiency may potentiate the diabetic state. Conn reported that primary aldosteronism exists in a state which is clinically indistinguishable from essential hypertension, in that the serum electrolytes are normal. They felt that it could be the cause of hypertension in as many as 20% of cases and from their own conservative estimate there are 3 million hypertensive patients with undiagnosed primary aldosteronism in the USA. The impaired carbohydrate tolerance they stated was possibly due to an inability of the beta cells to release insulin quickly in response to a rising blood sugar, and the defect is repairable by potassium supplementation. In the present study, potassium studies are not complete, but preliminary results have shown no difference in the diabetic as compared with the non-diabetic group. However Conn states: 'Among our total hypertensive population there is a fairly large number of persons who have mild to severe deficits of body potassium, whether or not this is reflected by the existing level of serum potassium.'

It will also be of interest to see if in the hypertensive group with an abnormal glucose-tolerance test, the GTT does revert back to normal with the use of potassium supplements.

In every hypertensive patient it is necessary to do a GTT, as this would affect the prognosis and therapy of the patient. Further studies similar to that of Conn will prove interesting in order to determine whether the abnormal GTT is reversible by the use of potassium supplements. The combination of diabetes and hypertension in the Natal Indian seems to lead to serious cardiac and cerebrovascular disease. Walker and Seftel stated that in Durban (214,000 Indians in 1959) cerebrovascular disease accounted for 23% of male and 22% of female deaths in the 55-74 age-group. Corresponding figures for Johannesburg Whites were 3.3% and 5.8% respectively.

McKechnie found that the incidence of abnormal GTT among 47 Natal Indians with hyperuricaemia was 59.6%. It is possible that hyperuricaemia, hypercholesterolaemia and coronary artery disease may be associated with hypertension and diabetes and that, as stated by McCollister, a fundamental biochemical abnormality may underlie these conditions.

**SUMMARY**

From this study there was a close correlation between hypertension and an abnormal glucose-tolerance test in the Natal Indian and African. There was a higher incidence of myocardial ischaemia in the Indian hypertensive diabetic compared with the African hypertensive diabetic and the Indian hypertensive non-diabetic. It would appear from the present study that the aetiology of hypertension and diabetes in the Natal Indian rests upon a biochemical factor.

I wish to thank Prof. A. J. Wilmot for his helpful criticisms and advice; Dr. E. R. Raine for his help at the Hypertension Clinic; the nurses at the Clinic; Dr. P. L. Patel, Department of Physiology, for his statistical assistance; and Dr. R. J. Wannenburg, the Medical Superintendent of King Edward VIII Hospital, for facilities.

**REFERENCES**


**WHITHER ELECTROCARDIOGRAPHY?**

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Proposals to change awkward century-old practices, like the use of non-decimal coinages, weights and measures into something more systematic, have usually been dismissed as being not only unnecessary, but unrealistic and economically insupportable. Moreover, such new-fangled ideas would be running counter to good usage, established custom and entrenched tradition, which had stood the test of time. What could be better?

Exactly similar arguments are being employed in favour of the retention of the universally used Einthoven system of electrocardiography. Here too we hear that it is good enough and what could be better?

During the past 10 years or so, electrophysiologists and medical scientists have however become progressively in-